Late stent fracture – A potential role of left ventricular dilatation

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Background: Coronary stent fracture is an under-recognized event but one that has been reported frequently in the drug-eluting stent era. Most reported cases of stent fracture occurred within days to two years after implantation, and are related to stent thrombosis and restenosis.

Case report: Presentation of a 69-year-old male with a history of arterial hypertension and previous percutaneous coronary intervention (PCI), and with implantation of three overlapping drug-eluting stents (DES) on proximal-to-middle left anterior descending artery (LAD). At five-year outpatient evaluation, the patient was found to have a new left bundle branch block associated with mild elevation in Troponin-I value and severe left ventricular dysfunction. The patient recovered as non ST-segment elevation myocardial infarction (NSTEMI) and consequently a new coronary angiography showed total occlusion of proximal LAD with multiple stent fracture. Here we discuss the role of left ventricular dilatation as a contributing factor to late drug-eluting stent fracture.

Conclusion: Different anatomical coronary settings have been described as predisposing factors to stent fracture. Consequently, the remodeling of the left ventricle, together with the rise in diastolic pressure, may have affected the shear stress of LAD stents by increasing mechanical forces produced in the diastolic phase on the epicardial vessel. In addition, left ventricular enlargement could have increased the elongation forces on the stent frames by altering the curvature of the stent. All predisposing factors of stent fracture, including coronary and left ventricular issues, need to be considered before stent implantation to avoid stent fracture and clinical sequelae.

Keywords: Drug-eluting stents (DES), Left ventricular remodeling, Percutaneous coronary intervention, Stent fracture

Introduction

Stent fracture (SF) after drug-eluting stents implantation has been reported in 1–8% [1,2] of cases, and is associated with stent thrombosis, myocardial infarction, and recurrent angina [2]. Predisposing factors for SF include: right coronary artery location, vessel tortuosity, overlapping and long stents, and stent implantation in angulated vessels (≥45°) and in saphenous

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vein grafts [2] and longitudinal stent distortion [5]. We report a case of multiple stent fracture causing total occlusion in the left anterior descending artery.

Case report

A 69-year-old male with history of arterial hypertension and previous percutaneous coronary intervention (PCI) on ramus with bare medical stent implantation presented to our department due to unstable angina. Coronary angiography revealed diffuse disease with multiple lesions in proximal and middle segments of left anterior descending artery (LAD). PCI was performed with implantation of three overlapping drug-eluting stents (DES) on proximal-to-middle LAD (Cypher 2.75/33 mm & 2.75/23 mm, Cordis Warren NJ, USA; plus Taxus 2.5 × 16 mm, Taxus® Boston Scientific Corp., Natick, MA) (Fig. 1). Subsequent yearly follow-up was uneventful until the fourth year. An outpatient evaluation at the fifth year revealed new left bundle branch block associated with mild elevation in Troponin-I value (TnI 0.053 ug/L) and severe left ventricular dysfunction (EF 25%). At that time, the patient was free of symptoms. A new coronary angiography showed total occlusion of proximal LAD with multiple stent fracture (type V). The fracture was best visible in fluoroscopy without contrast injection, clearly showing acquired transection and discrete gap in the stent body (type V) (Fig. 2). Unfortunately, the patient refused any further intervention for revascularization or any other evaluation such as implantable cardioverter/defibrillator. He was thus discharged on medical therapy only.

Discussion

Coronary stent fracture is an under-recognized event but one that has become more frequent in the DES era. Most reported cases of stent fracture

Figure 1. Baseline coronary angiogram (2005) in the setting of unstable angina, showing multiple tight stenosis on LAD (arrows) (a) AP cranial view, and (b) lateral view. Post intervention angiogram showing angiographic success after three overlapping drug-eluting stents (c). Coronary angiography demonstrating no disruption of stent struts (d) RAO cranial view, and (e) lateral view. Left ventricular angiography revealed moderate left ventricular dysfunction (f).
have occurred within days to two years after implantation [3]. Studies report multiple factors contributing to stent fracture. The majority of fractured coronary stents described in the literature occur in the right coronary artery (RCA). The forceful and exaggerated motion of the RCA in the atrioventricular groove during the cardiac cycle has been associated with stent fractures [4]. Vein grafts to the RCA may also be subject to high mechanical stress because they curve at the acute margin of the heart for insertion into the native vessel [3]. Stent length has been associated with stent fracture, possibly because longer stents are subject to higher radial forces, especially in their mid segment [4]. Fractures have also been reported in ostial lesions, at the point of the maximal vessel curvature in tortuous vessels, in calcified lesions, and at regions of stent overlap at which each extremity may act as a fulcrum resulting in metal deformation, and increased rigidity of overlapping stents, as in our case [4]. Furthermore, stent overexpansion may weaken the stent struts and promote fracture [3]. The broken struts cause local mechanical stimulation of the vessel wall, resulting in inflammation and mobilization of the intimal hyperplasia mechanism. In addition, the fracture leads to the destruction of the stent architecture locally, so that the drug, which is distributed evenly along its length, is no longer present at a particular location [4]. In addition, there may be significant malapposition of struts along with disrupted flow that may cause predisposition to stent thrombosis [5]. In our case, the stent length as well as the presence of multiple overlapping stents, and stent overexpansion may have lead to stent fracture and subsequent vessel occlusion, related to stent thrombosis or occlusive restenosis, with left ventricular failure and dilatation. However, we also thought that left ventricle dilatation and increased left ventricular end-diastolic pressure, due to chronic ischemic disease, increasing stent frames shear stress, might have contributed to transection of stent frames. In fact, studies posit that stent fractures may also result from excessive mechanical vessel wall stress that occurs from extreme repetitive contraction and flexion of the vessel during cardiac cycles [4]. As a result, the remodeling of the left ventricle together with the rise in diastolic pressure, as observed in our case, may have affected the shear stress of LAD stents, by increasing mechanical forces produced in the diastolic phase on the epicardial vessel. In addition, left ventricular enlargement could have increased the

Figure 2. Follow-up coronary angiogram (2012). Total occlusion intra-stent of mid-LAD (a) RAO cranial view, and (b) lateral view. Still-frame angiography showing multiple SF of LAD (type V) with acquired transection with gap in the stent body, fracture sites are pointed by arrows (c) AP cranial view, (d) RAO cranial view, and (e) lateral view. Left ventricular angiography revealed severe LV dysfunction (f).
elongation forces on the stent frames by altering the curvature of the stent. In a recent meta-analysis on drug-eluting stent fracture, Chakravarty et al. found that stent strut fracture was more common in sirolimus-eluting stents compared to paclitaxel-eluting stents [4]. This difference has been also associated with the closed-cell design of sirolimus-eluting stents, characterized by larger numbers of connectors between hoops, compared to the open-cell design of paclitaxel-eluting stents. In fact, research has illustrated that stents with two connectors between hoops have less longitudinal strength when exposed to compressing or elongating forces than those with more connectors [5]. Thus, the latter are more resistant to shortening or elongation, resulting in a more rigid structure prone to fractures [5]. Diagnosis of stent fracture relies mainly on coronary angiography or intravascular ultrasound (IVUS). The true incidence may be under-reported as follow-up angiogram is not routinely adopted. Indeed, IVUS may be a more sensitive method for the detection of stent fracture [6]. IVUS was not performed in our case because of total occlusion of coronary vessel. However, in this case, angiography depicted very distinctive images of circumferential stent fracture.

Conclusions

Coronary stent fracture is an under-recognized event which has been reported more frequently in the DES era. Most reported cases of stent fracture have occurred within days to two years after implantation, and involve stent thrombosis and restenosis. Different anatomical coronary settings have been described as predisposing factors to stent fracture. We discussed the role of left ventricular dilatation as a contributor factor to late drug-eluting stent fracture. In order to avoid stent fracture and its clinical sequelae before stent implantation, all predisposing factors of stent fracture, including coronary and left ventricular issues, should be considered.

References